Assessing right ventricular function: the role of echocardiography and complementary technologies

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The physiological importance of the right ventricle (RV) has been underestimated; the RV was considered mainly as a conduit whereas its contractile performance was thought to be haemodynamically unimportant.[1] However, its essential contribution to normal cardiac pump function is well established with the primary RV functions being:

- to maintain adequate pulmonary perfusion pressure under varying circulatory and loading conditions in order to deliver desaturated venous blood to the gas exchange membranes of the lungs
- to maintain a low systemic venous pressure to prevent tissue and organ congestion.

RV function may be impaired either by primary right sided heart disease, or secondary to left sided cardiomyopathy or valvar heart disease.[2] In addition, it should be considered that RV dysfunction may affect left ventricular (LV) function, not only by limiting LV preload, but also by adverse systolic and diastolic interaction via the intraventricular septum and the pericardium (ventricular interdependence). Moreover, RV function has been shown to be a major determinant of clinical outcome[3–9] and consequently should be considered during clinical management and treatment.[10] Thus, the need for diagnosis of RV dysfunction is evident. In practice, clinicians largely rely on non-invasive imaging methods for assessment of RV function. Two dimensional echocardiography is the mainstay for analysis of RV function, but recently alternative techniques have been proposed, including tissue Doppler imaging (TDI) techniques,[11] three dimensional echocardiography,[12] magnetic resonance imaging (MRI), and even invasive assessment of pressure–volume loops.[13–17] An overview of these imaging modalities for assessment of RV function is provided in the current manuscript.

ECHOCARDIOGRAPHIC EVALUATION OF THE RIGHT VENTRICLE

Due to its widespread availability, echocardiography is used as the first line imaging modality for assessment of RV size and RV function. The quantitative assessment of RV size and function is often difficult, because of the complex anatomy. Nevertheless, when used in a qualitative fashion, two dimensional echocardiography can easily obtain valuable information about RV size and function.

Two dimensional echocardiography

For *qualitative* evaluation, the RV size should be compared to the LV size. In the parasternal long axis and apical four chamber views, the normal RV is approximately two thirds the size of the LV. If the RV appears larger than the LV and/or shares the apex, RV dilatation may be present. Confirmation in other views is needed to avoid false positive findings. From short axis projections, the RV should be smaller than the LV while the LV shape should have a circular geometry throughout the cardiac cycle. Finally, the RV should also be evaluated from the subcostal projections. If the RV appears larger in length or diameter, RV dilatation is likely to be present.[18] RV size (end systolic and end diastolic) and change in size during the cardiac cycle (RV function) can also be *quantitatively* assessed by tracing the RV endocardial border or measuring RV dimensions. However, this is often cumbersome and interobserver variability is high. A wide variety of techniques have been proposed, but none is currently considered as the gold standard.[4 19–30] Studies using endocardial tracing of the RV area report relatively high correlations (0.69–0.88) between echocardiographically estimated RV size and function compared to radionuclide angiography and MRI.[21 22 26] However, the number of patients who could not be analysed because of failure to trace the (entire) RV myocardium is large[21 22 27]; RV tracing may be improved using intravenous contrast agents, that are commercially available (fig 1). The most widely used quantitative technique is the area–length method in which a traced RV lumen area in the four chamber view is combined with the RV dimension in the parasternal short axis view.[24 25 28 29] A different quantitative approach to assess RV function is the measurement of the tricuspid annular plane systolic excursion (TAPSE). The TAPSE estimates RV systolic function by measuring the level of systolic excursion of the lateral tricuspid valve annulus towards the apex in the four chamber view (fig 2). An excellent correlation between the TAPSE and

Figure 1 Subcostal projections without (left) and with (right) intravenous contrast injection (SonoView). Notice the clear outline of the right ventricular (RV) endocardial definition together with the RV trabeculations.

Abbreviations: LV, left ventricular, MRI, magnetic resonance imaging; RV, right ventricular; RT3DE, real time three dimensional echocardiography; TAPSE, tricuspid annular plane systolic excursion; TDI, tissue Doppler imaging

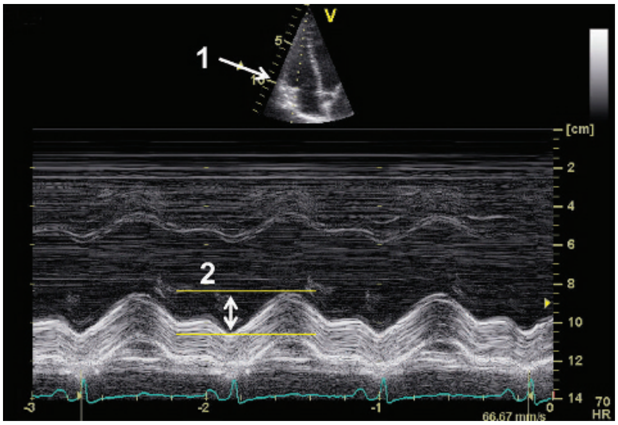


Figure 2 Tricuspid annular plane systolic excursion (TAPSE) in a normal individual. In the four chamber view a straight line (M mode) is drawn through the lateral tricuspid valve annulus (arrow 1). The level of excursion of the tricuspid valvar plane during systole (TAPSE, in mm) corresponds with RV ejection fraction (arrow 2) (5 mm ~ 20% RV ejection fraction, 10 mm ~ 30% RV ejection fraction, 15 mm ~ 40% RV ejection fraction, and 20 mm ~ 50% RV ejection fraction).[21]

RV ejection fraction as assessed by radionuclide angiography was shown.[21] The approach appears reproducible and proved to be a strong predictor of prognosis in heart failure.[21,23,31]

The Doppler index of myocardial performance (Tei index or myocardial performance index) is yet another parameter that can be used for evaluation of RV performance.[32] It is expressed by the formula [(isovolumic contraction time + isovolumic relaxation time)/RV ejection time] (fig 3). It is established that is actually unaffected by heart rate, loading conditions or the presence and the severity of tricuspid regurgitation.[33]

Tissue Doppler imaging

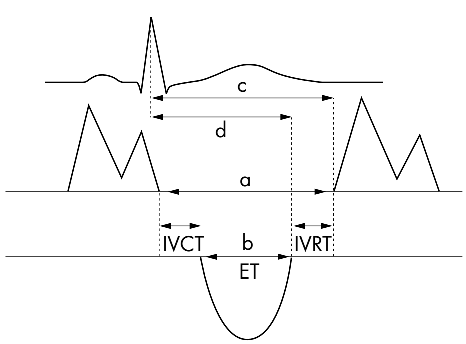
TDI allows quantitative assessment of RV systolic and diastolic function by means of measurement of myocardial velocities. The earlier studies used pulsed wave TDI to examine RV function (fig 4). Two dimensional colour coded TDI, however, allows true offline analysis of multiple segments simultaneously (fig 5). Peak systolic velocity < 11.5 cm/s identifies the presence of RV dysfunction with a sensitivity and specificity of 90% and 85%, respectively.[34]

In patients with inferior myocardial infarction and RV involvement, the tricuspid lateral annular systolic and early diastolic velocities were significantly reduced when compared to healthy individuals and patients without RV involvement.[35,36] In heart failure patients, the reduction of tricuspid annular systolic velocity is associated with the severity of RV dysfunction.[37] Moreover, non-invasive estimation of right atrial pressure is possible using trans-tricuspid pulsed wave Doppler and TDI (E/E′, right atrial pressure = 1.76 (E/E′) – 3.7).[38] In hypertrophic cardiomyopathy, subclinical involvement of the RV is also evident by a reduction of tricuspid annular peak systolic and early diastolic velocities and reversal of tricuspid annulus E′/A′ ratio.[39] Still, clinical experience in patients is limited and further studies with TDI assessing RV function are needed.

Besides assessment of RV function, TDI does also permit assessment of ventricular dyssynchrony. This has been extensively demonstrated in the LV, but the prevalence and haemodynamic consequences of RV dyssynchrony in cardiac disease are not well defined. Theoretically, RV dyssynchrony may affect the coordination of contraction in different RV regions resulting in reduced cardiac output. However, this phenomenon may also be mediated by the change in movement of the interventricular septum. Currently, the number of RV segments that can be assessed by TDI is limited, and only measurement of septum-to-RV free wall dyssynchrony is feasible.[40] In heart failure patients with a prolonged QRS duration on the ECG, septum-to-RV free wall dyssynchrony has been observed, which was reversed after cardiac resynchronisation therapy.[40] Also, RV dyssynchrony has been suggested to predict response to cardiac resynchronisation therapy,[41] although other studies did not confirm this observation.[42] Also, it is not clear whether patients with isolated RV dyssynchrony will benefit from cardiac resynchronisation therapy. In general, studies are needed to understand better the clinical meaning of RV dyssynchrony in cardiac disease.

Strain rate imaging

While the assessment of *longitudinal* strain from the apical views is feasible in the clinical setting, the analysis of RV *radial* deformation from the parasternal window turned out to be difficult. It is hampered by near-field artefacts caused by the close proximity to the transducer and by the thin wall thickness, which requires an extremely small computational distance of less than 5 mm for strain rate measurements.[4,37,39,43–51] In healthy individuals, RV longitudinal velocities demonstrated the typical baso-apical gradient with higher velocities at the base; also, RV velocities are consistently higher as compared to the LV.[43,44] This can be best explained by: (1) the differences in loading conditions and compliance with a lower afterload in the RV; and (2) the dominance of longitudinal and oblique myocardial fibres in the RV free wall.[52] In contrast to the homogenously distributed deformation properties within the LV, the strain rate and strain values are more inhomogeneously distributed in the RV and show a reverse baso-apical gradient, reaching the highest values in the apical segments and outflow tract.[43,51] This pattern can be best explained by the complex geometry of the thin-walled, crescent shaped RV and the more inhomogenous distribution of regional wall stress if compared to the thick-walled, bullet shaped LV. In an elegant animal experiment, Jamal *et al*[51] compared echocardiographic strain rate imaging results to sonomicrometry and demonstrated the feasibility of the echo technique to quantify changes in RV contractile function. Doppler derived strain measurements correlated well to sonomicrometry segment length measurements both in the inflow and outflow tract of the RV and under different loading conditions. An acute increase in RV afterload led to an increase in RV myocardial strain rate, a measure of contractile function,[53] and to a



$$\text{Index} = \frac{a - b}{b} = \frac{(\text{IVCT} + \text{IVRT})}{\text{ET}}$$

Figure 3 Schematic display of pulsed wave Doppler recordings from RV inflow and outflow tract projections demonstrating the calculation of Myocardial Performance Index (Tei index). ET, ejection time; IVCT, isovolumic contraction time; IVRT, isovolumic relaxation time.

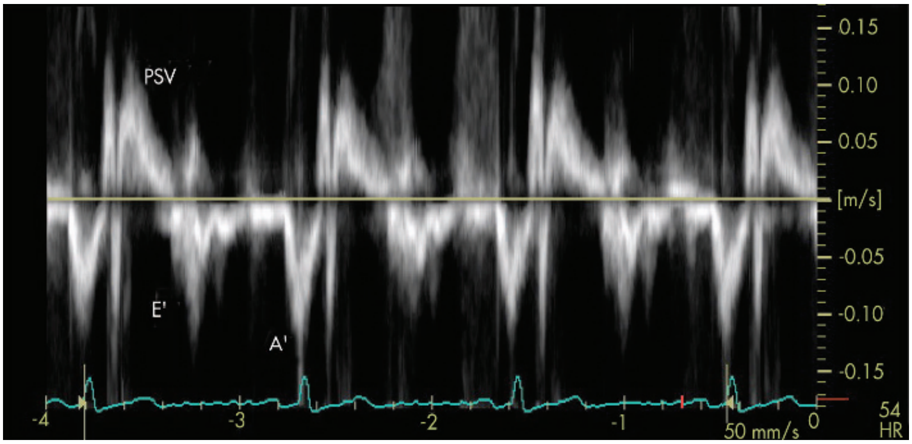


Figure 4 Tracing derived from pulsed wave tissue Doppler with the sample placed at the level of the tricuspid annulus of the RV free wall, demonstrating peak systolic velocity (PSV), and diastolic parameters (E′ and A′).

decrease in peak systolic strain, indicating a decrease in RV stroke volume. Importantly, not only the absolute values changed, but also the strain profile after pulmonary artery constriction demonstrated a shift of myocardial shortening from early-mid to end systole or even early diastole (post-systolic shortening).[51]

Dambrauskaite *et al*[50] published a case study on the changes in regional RV myocardial function after bilateral lung transplantation in a patient with primary pulmonary hypertension. Conventional echocardiography showed a significant improvement in RV size and global function after successful transplantation, but strain rate imaging revealed that the functional improvement was limited to the apical, trabecularised portion of the RV and that the smooth inlet segment did not improve after afterload reduction. Before transplantation, peak systolic strain in the apical segment was significantly delayed and occurred in the early diastolic phase after tricuspid valve opening. After transplantation with afterload reduction, it was shifted towards the systolic ejection period and occurred even before pulmonary valve opening, thus confirming the experimental findings by Jamal *et al*.[51] Furthermore, preliminary data in patients with pulmonary hypertension suggested that in a compensated patient, peak systolic strain rate correlated with peak systolic pulmonary artery pressure and that regional function will first exhibit depression in the smooth inlet portion of the RV.[54] In this setting, regional analysis of myocardial function may enable the early diagnosis of imminent RV failure before irreversible damage will occur.

In summary, the available experience on strain rate imaging for the assessment of RV function is limited to small single centre studies and case reports. The technique seems feasible for the quantitative assessment of RV function and may improve understanding of the pathophysiology of different diseases. However, the clinical value for patient management remains to be proven.

Three dimensional echocardiography

The clinical use of three dimensional echocardiography has been hindered by the prolonged and tedious nature of data acquisition. The recent introduction of real time three dimensional echocardiography (RT3DE) has revolutionised echocardiography as images may be obtained in just one beat. This has been achieved by the development of a full matrix array transducer (X4, Philips Medical Systems, Andover, Massachusetts, USA), which utilises 3000 elements. This has resulted in (1) improved image resolution, (2) higher penetration and (3) harmonic capabilities, that may be used

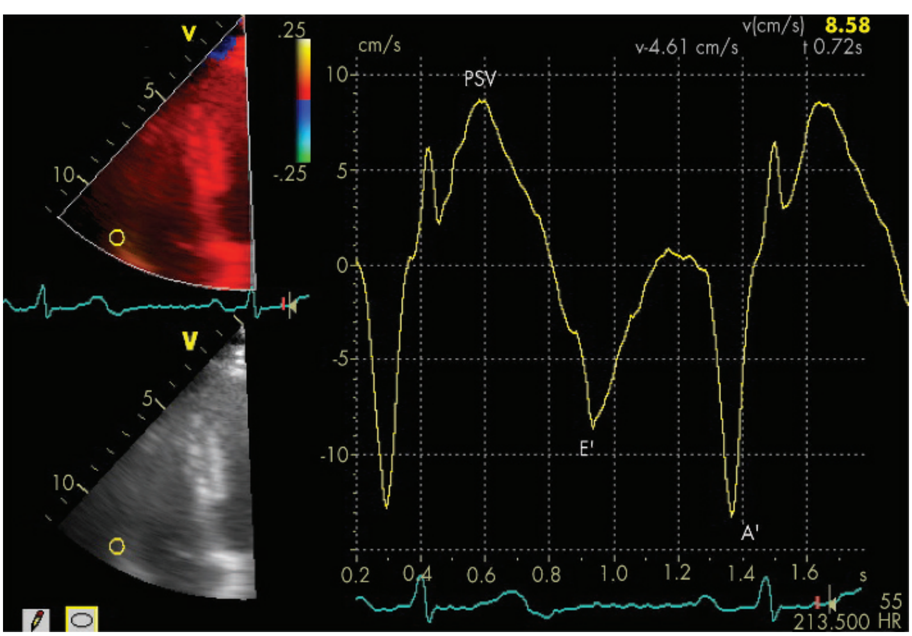


Figure 5 Tracing derived from colour coded tissue Doppler imaging with the sample placed at the level of the tricuspid annulus of the RV free wall, demonstrating peak systolic velocity (PSV), and diastolic velocities (E′ and A′).

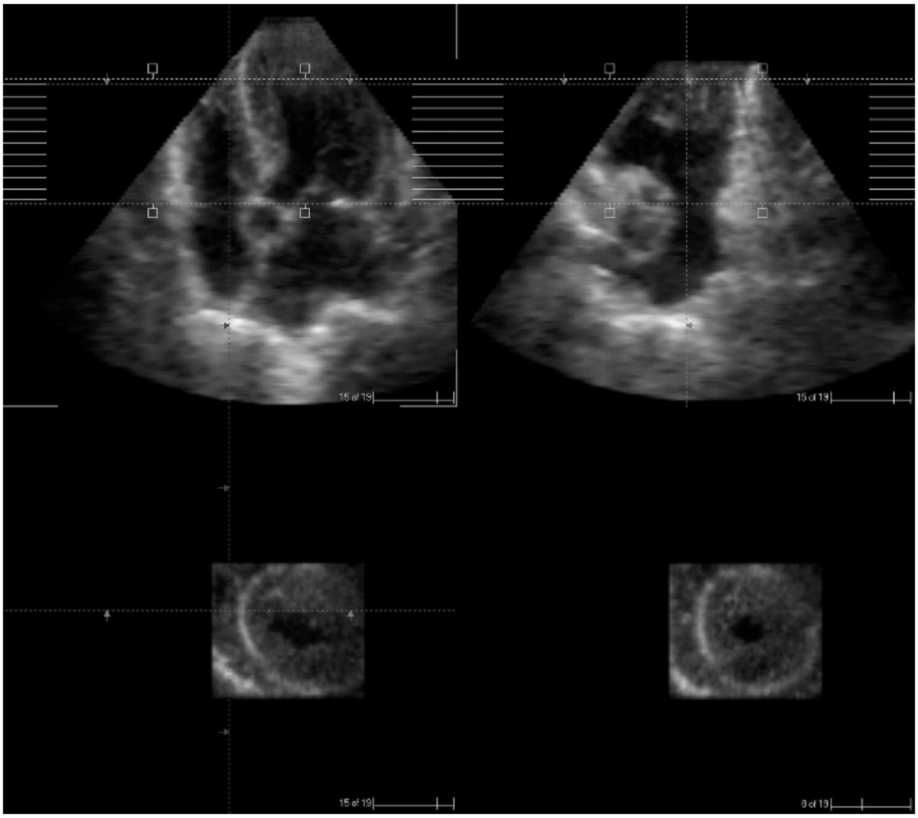


Figure 6 Three dimensional acquisition of the right ventricle using an automated disk algorithm for volume calculations.

for both grey scale and contrast imaging. In addition, this transducer displays "on-line" three dimensional volume rendered images and is also capable of displaying two simultaneous orthogonal two dimensional imaging planes.

The major advantage of RT3DE is that volumetric analysis does not rely on geometric assumptions, as has been the case with two dimensional echocardiography. Quantification of LV volumes and mass using RT3DE has successfully been performed from an apical wide angled acquisition using different methods. A similar approach can be applied for RV evaluation. Data analysis may be performed on-line or off-line with dedicated three dimensional software (4D LV analysis, TomTec GMBH, Munich, Germany) (fig 6). Since a data set comprises the entire RV volume, multiple slices may be obtained from the base to the apex of the heart as in the method of discs. This acquisition can then be combined with intravenous contrast agents to improve endocardial border delineation and RV end diastolic and end systolic volumes can be calculated by tracing the endocardial borders similar to MRI (figs 7 and 8).

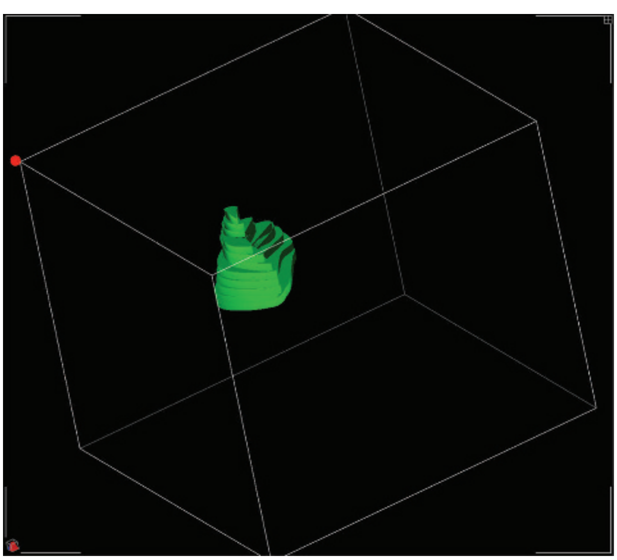


Figure 7 Three dimensional representation of end diastolic right ventricular volume.

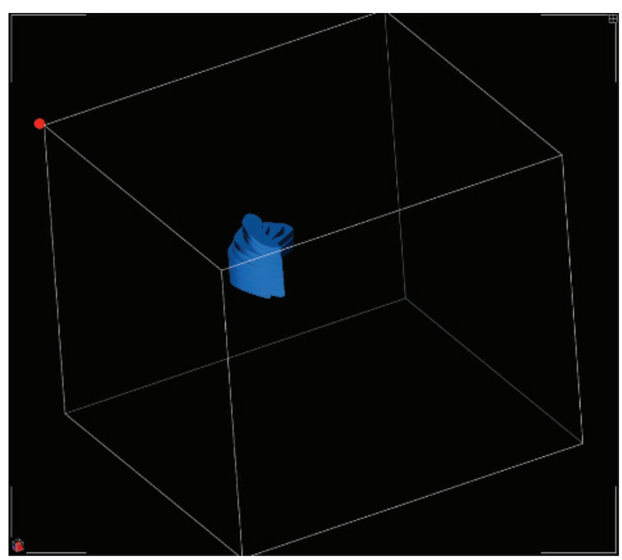


Figure 8 Three dimensional representation of end systolic right ventricular volume.

MRI TO ASSESS RIGHT VENTRICULAR FUNCTION

In recent years, MRI scanners and imaging protocols have developed rapidly. At present, imaging is generally performed on 1.5 Tesla systems, using dedicated cardiac phased-array coils with multiple elements and ECG triggering. Optimal results are obtained using fast breath-hold techniques, echo-planar or balanced fast field echo. Mogelvang *et al*[55] demonstrated the accuracy of MRI to assess RV volumes. The reproducibility of the technique was shown by Grothues *et al*[56] who evaluated 60 individuals (20 healthy subjects, 20 heart failure patients, 20 patients with ventricular hypertrophy) on two different occasions. The authors demonstrated an excellent reproducibility for assessment of RV function and RV volumes using MRI. Functional (and anatomical) images of both the LV and RV are commonly obtained in the short axis direction. Alfakih and colleagues[57] compared RV volume measurements in the short axis and in axial directions. The axial orientation resulted in a better intra- and inter-observer reproducibility and may be considered as the preferred direction for assessment of RV function.

MRI can also be used for measurement of flow velocity and volume by phase velocity mapping. Phase velocity mapping is based on gradient-echo pulse sequences in combination with ECG triggering. The phase contrast allows velocity encoding and therefore flow measurements.

Contrast enhanced MRI for imaging of myocardial scar tissue was first described more than 20 years ago.[58 59] With an inversion recovery turbo field echo pulse, a heavily T1 weighted image is obtained that maximises the contrast between the scarred (dead) and normal myocardium; accordingly scarred myocardium appears bright whereas normal myocardium is dark. Recent studies have reported excellent correlations between scar tissue on MRI and postmortem analysis of infarcted myocardium.[60] The majority of studies focused on assessment of scar tissue in the LV, but Sato and colleagues[61] demonstrated the feasibility of MRI for assessment of scar tissue in the RV. An example of contrast enhanced MRI to assess RV scar formation in a patient with inferior infarction with RV involvement is shown in fig 9.

Despite the excellent image quality and reproducibility, MRI has some disadvantages: the data acquisition and analysis is rather time consuming, and some patients groups (for example, pacemaker patients) cannot undergo MRI. Still, MRI can currently be considered the most accurate method for assessment of RV size and function and may be well suited for quantification of RV scar tissue following infarction.

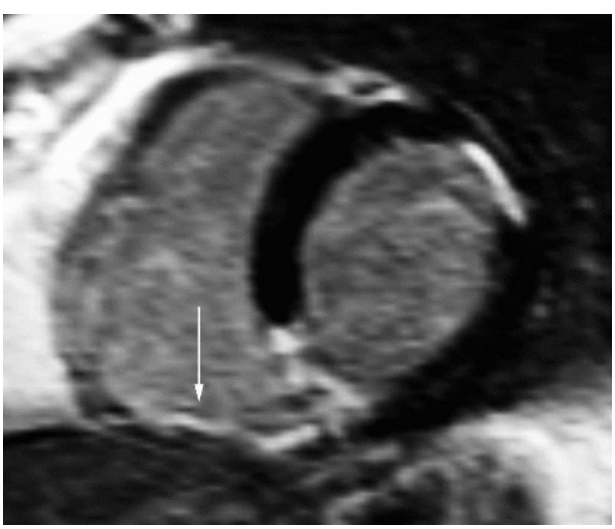


Figure 9 Contrast enhanced magnetic resonance image; short axis view showing right ventricular scar tissue in a patient with acute inferior infarction (arrow). A second infarcted area is present in the anterolateral region of the left ventricle.

PRESSURE–VOLUME LOOPS TO ASSESS RIGHT VENTRICULAR FUNCTION

Analysis of RV function by pressure–volume loops is attractive because it quantifies various determinants of ventricular function in a relatively independent fashion.[62–64] Figure 10 depicts typical steady state RV pressure–volume signals and the corresponding pressure–volume loops. Conventional indexes such as end systolic and end diastolic pressures and volumes, stroke volume, stroke work and ejection fraction can be directly derived from the loops. Furthermore, differentiation of the volume signal (dV/dt) yields RV in- and outflow signals, from which early and late filling rates and ejection rate can be obtained. Analysis of the pressure decay during relaxation yields the relaxation time constant τ, whereas differentiation of the pressure signal yields dP/dt_{MIN} and dP/dt_{MAX}. These indexes reveal important information on systolic and diastolic RV function, but it should be noted that they not only depend on intrinsic RV function, but are also affected by loading conditions. This is clearly illustrated in fig 11, which shows pressure–volume loops obtained during a preload reduction. In contrast, the pressure–volume *relations*, which may be derived from the pressure–volume loops during the loading intervention, provide indexes of RV function that are largely independent of loading conditions and thus more specifically reflect intrinsic myocardial function. The position and slope (end systolic elastance) of the end systolic pressure–volume relation are sensitive indicators of RV systolic function. Alternative indexes are the slope of the relation between stroke work and end diastolic volume, the preload recruitable stroke work, and the slope of the relation between dP/dt_{MAX} and end diastolic volume.[65] Diastolic function is derived from the end diastolic pressure–volume points. The linear slope of this relation represents diastolic stiffness or, more commonly, diastolic compliance (1/stiffness). When obtained over a wider range, the end diastolic pressure–volume relation is generally non-linear and better approximated by an exponential fit, such as $EDP = A \cdot \exp(k \cdot EDV)$ and characterised by the diastolic stiffness constant (k).[16]

Construction of pressure–volume relations requires pressure–volume loops obtained during various loading conditions, preferably induced by interventions that minimally affect intrinsic myocardial function. An elegant means to achieve this is temporary balloon occlusion of the inferior vena cava. This procedure enables a rapid, purely mechanical, reduction in preload, and prevents reflex mechanisms. Alternatively, methods have been proposed that provide estimates of systolic pressure–volume relations based on steady state pressure–volume loops.[66] Likewise, diastolic compliance may be estimated by considering multiple pressure–volume points during the late filling phase. These single-beat approaches are attractive because they avoid the need for a loading intervention, but the accuracy of the derived indexes especially in the diseased RV needs further study.

Currently, the conductance catheter is the most frequently used instrument to assess RV pressure–volume loops.[67] This catheter contains a high fidelity pressure sensor, and up to 12 electrodes to measure RV electrical conductance from which instantaneous RV cavity volume is determined.[68] Pressure–volume loop analysis is extensively used in experimental studies[14 17 69–73] and is generally considered the optimal way to quantify RV function. In patients, this approach has been validated[15] and used to assess RV function in congenital heart

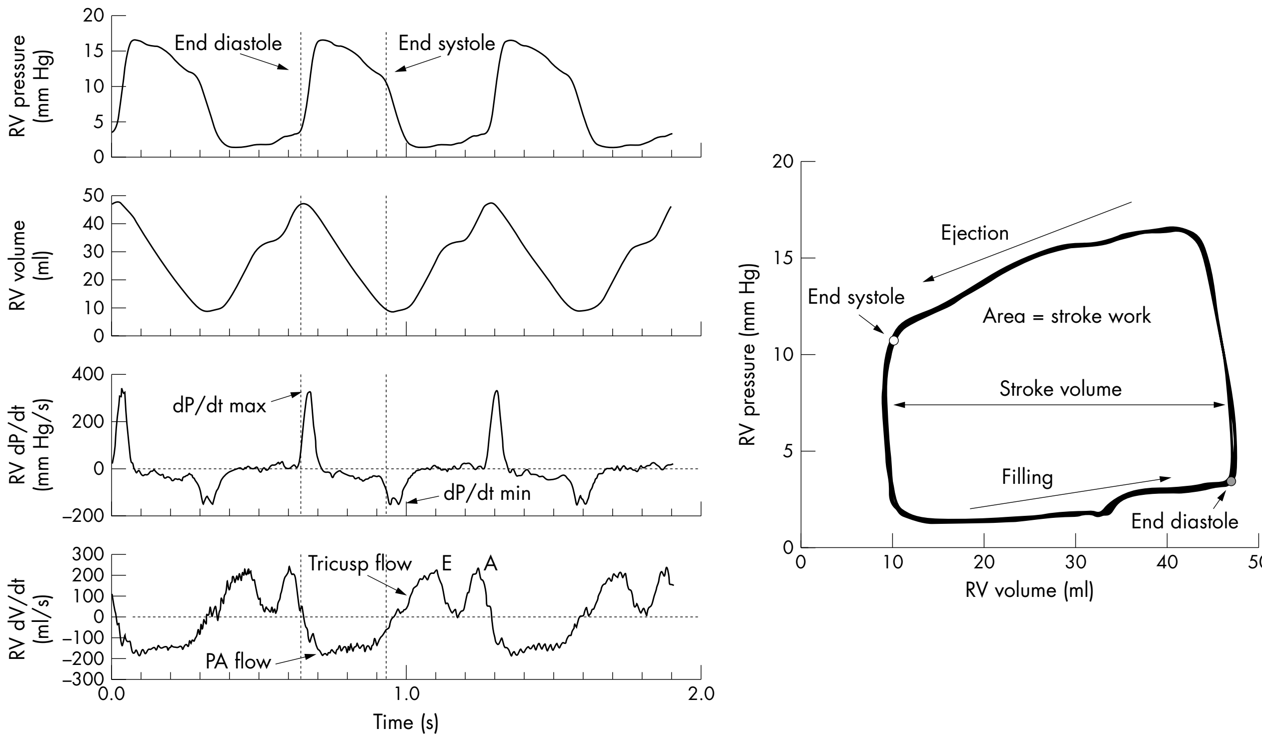


Figure 10 Steady state right ventricular (RV) pressure–volume signals and pressure–volume loops obtained by conductance catheter in a 25 kg sheep.

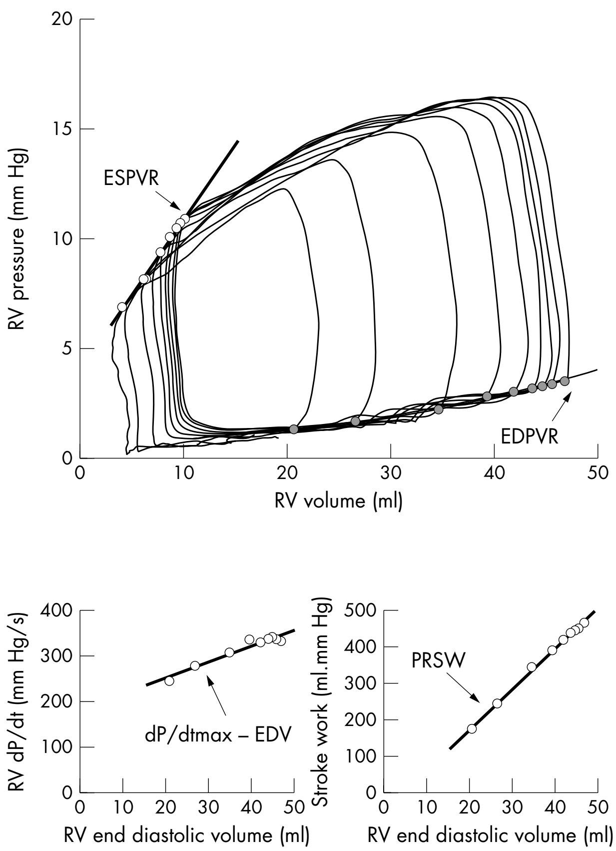


Figure 11 Right ventricular (RV) pressure–volume loops during a loading intervention (preload reduction by gradual vena cava occlusion) and derived pressure–volume relations. dP/dtmax – EDV, dP/dt_{MAX} – end diastolic volume relation; EDPVR, end diastolic pressure–volume relation; ESPVR, end systolic pressure–volume relation; PRSW, preload recruitable stroke work relation.

disease[74] and postoperative RV function.[15] In summary, it is concluded that pressure–volume analysis of RV function in patients is feasible but requires invasive measurements. Importantly, pressure–volume loops provide load-independent indexes that enable accurate diagnosis of systolic and diastolic RV function.

CONCLUSION

RV function is an important parameter in cardiac disease. In the clinical arena, two dimensional echocardiography can be used to assess RV dysfunction. Several new echocardiographic techniques, including TDI, strain rate imaging, RT3DE and contrast echocardiography, may further enhance our capability of assessing RV function. MRI is highly accurate for the assessment of RV function; however, availability is still limited and data analysis is time consuming. Finally, RV function can be evaluated invasively using pressure–volume loop analysis, which has the advantage of being relatively load independent.

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